

APPENDIX A

LITERATURE REVIEW

BACTERIAL WATERBORNE OUTBREAKS

Drinking Water

Drinking water-related outbreaks of pathogenic *Escherichia coli* have been reported as early as 1965 (in Sweden) and 1971 (in the United States) ¹. Since then, drinking water-related outbreaks of pathogenic *E. coli* have occurred throughout the world, including the United States ^{2, 3, 4, 5, 6}, the United Kingdom ^{7, 8}, the Caribbean ⁹, the Canary Islands ¹⁰, and Japan ¹¹. Verotoxigenic *Escherichai coli* (VTEC) have been implicated in nine of these outbreaks: seven outbreaks involved serotype O157:H7 ^{2, 3, 4, 11, 5, 10}, one outbreak involved serotype O111:H8 ⁶, and one involved verotoxin producing *E. coli* serogroup O157 ⁸. Three outbreaks of enterotoxigenic *E. coli* (ETEC) on cruise ships in the Caribbean involved multiple serotypes including O169:H41, O148:H28, and O8:H9 ⁹. In total, two outbreaks have occurred in municipal systems. Nine outbreaks have occurred in private water supplies, including three cruise ships, a trailer park, two camps, and a school. One outbreak appears to be due to contamination of the water after leaving the distribution system.

Five of the nine VTEC outbreaks are attributed to contamination of the water source. In three outbreaks, the drinking water source was unchlorinated groundwater, and it was probable that the wells or springs were contaminated by livestock or wildlife grazing nearby ^{4, 10, 8}. One of the outbreaks occurred at a campsite in Scotland and was linked to a private water supply⁸. It is noteworthy that visitors to the campsite became ill, but the local population sharing the same supply did not. In two outbreaks, the drinking water source was unchlorinated or improperly chlorinated groundwater which was determined to be under the influence of surface water ^{3, 5}.

The largest of these outbreaks involved a municipal system and occurred in Alpine, Wyoming in the summer of 1998 ⁵. Factors contributing to the outbreak included a groundwater source under direct influence of surface water, potential cross-connections, and weather and geology. In this outbreak, a total of 114 cases of diarrhea, four cases of hemolytic-uremic syndrome (HUS), and no deaths were reported. It is noteworthy that several visitors to the Alpine area became ill after only one exposure to Alpine water.

One of the nine VTEC outbreaks is attributed to contamination of water within the distribution system ². Sewage contamination of the municipal water distribution system during repair work was the probable cause of the outbreak of VTEC in Cabool, Missouri, in December 1989 – January 1990. This was the first documented waterborne outbreak of VTEC, and the largest outbreak of VTEC at the time. The outbreak resulted in 243 cases, 86 with bloody stools, 32 hospitalizations, four cases of HUS, and two deaths. One visiting case was in town for two hours and drank one glass of water before becoming ill.

Of the remaining three VTEC outbreaks, one was attributed to poor kitchen sanitation and contamination of ice ⁶. Information on the other two outbreaks is limited, however, one outbreak resulted from a chlorinated groundwater system that did not comply with regulations ⁴, and the other from a contaminated well at a school ¹¹.

The three EHEC outbreaks occurred on cruise ships travelling along the Atlantic coast of North America and in the Caribbean⁹. Inadequate chlorination contributed to two of the three outbreaks. Water bunkered by cruise ships in ports outside of the United States, and ice made from that water, were the implicated vehicles.

The earliest documented drinking water-related outbreak of *Campylobacter* spp. occurred in Vermont in 1978¹², in a municipal system in which the water was inadequately chlorinated. Since then, drinking water-related outbreaks of *Campylobacter* spp. have been reported throughout Canada^{13, 14, 15, 16}, the United States¹⁷, the United Kingdom^{18, 19, 20, 21, 22, 23}, Finland²⁴, Norway^{25, 26}, Sweden²⁷, Australia²⁸, and New Zealand^{29, 30}. In total, nine outbreaks have occurred in municipal systems and twelve have occurred in private supplies, including a hospital and a resort.

Eleven outbreaks have occurred in a groundwater source. Of these, nine have occurred in systems without chlorination^{29, 18, 19, 20, 21, 16, 23, 27}, and two in systems where the chlorination was inadequate^{17, 24}. One of the largest of these outbreaks occurred in Orangeville, Ontario, in April 1985¹⁶. Heavy spring runoff and meltwater contamination of the municipal wells combined with lack of chlorination in the system was the probable cause of the outbreak. This outbreak reported 241 cases, of which 57 were confirmed. Another outbreak in Greenville, Florida, in May 1983, involved a chlorinated municipal well water system with an open top settling tank, which was likely contaminated with feces from roosting birds¹⁷. A total of 79 cases were identified in the sample taken from the community, leading to an estimated 865 cases in total.

Nine outbreaks have occurred in systems where the source was surface water. Of these, five have occurred in systems without chlorination^{25, 26, 14, 19, 15, 21}, and four have occurred in systems where chlorination was inadequate^{13, 30, 19, 21, 22}. The largest of these occurred in a municipal system in Norway, in the summer of 1984^{25, 26}. Unchlorinated water from an unfenced reservoir was flooded by heavy rains and was probably contaminated by feces from sheep grazing in the area. A total of 680 cases occurred.

One outbreak was associated with the consumption of contaminated rainwater. At an island resort in North Queensland, Australia, in June 1997, water drawn from raintanks contaminated with animal feces was consumed by the staff of the resort²⁸. A total of 23 cases among the over 600 staff members occurred.

Two drinking water-related outbreaks involving both *E. coli* O157:H7 and *Campylobacter* spp. have been reported to date. The first occurred in Fife, Scotland, in March 1995³¹. Stream water into which human sewage was discharged contaminated the municipal water supply of a village in Fife for a period of five days. A total of 633 cases were identified, including eight confirmed *Campylobacter* spp. and six confirmed verotoxin-producing *E. coli* O157. Two cases of HUS and no deaths were reported.

The second outbreak occurred at the Washington County Fair, in New York State, in August 1999^{32, 33}. A shallow well supplying unchlorinated water to several vendors for beverages and ice was considered under the influence of surface water and was likely contaminated by manure. Of the 108,000 attendees, 921 cases were identified, including 116 confirmed *E. coli* O157:H7, 32 confirmed *Campylobacter jejuni*, and 13 co-infections. 65 hospitalisations, 11 cases of HUS, and two deaths were reported.

Other bacterial drinking water-related outbreaks have occurred, involving *Salmonella typhimurium*³⁴, *Shigella sonnei*^{3, 35, 6, 36}, and *Plesiomonas shigelloides* together with *Salmonella* Hartford³⁷. Contamination of the water with human sewage^{35, 36}, contamination of the water with bird or livestock feces^{34, 37}, and inadequate chlorination^{3, 34, 37} were cited as contributing factors. Two of these outbreaks occurred in municipal systems and four in private systems, including a resort and a local fair.

Recreational Water

Outbreaks attributed to recreational water exposure have been documented. VTEC infection has been linked to a poorly chlorinated private pool³⁸, to a beach³⁹, and to swimming in lake water^{40, 41, 42, 43}. In five of the six outbreaks^{40, 41, 38, 42, 39}, contamination of the water with human feces was the probable source of the outbreak, and in two of the outbreaks human feces was observed in the water^{40, 41}. Contamination of the water with animal feces was also mentioned as a possible source for two of the outbreaks^{41, 39}. A common factor among all six outbreaks is that all water was unchlorinated.

OCCURRENCE IN ONTARIO AND THE RELATION TO HUMAN DISEASE

Farm Family Studies

A prospective study of 80 dairy farm families in Ontario was conducted to examine the transmission of VTEC from cattle to humans^{44, 45}. *E. coli* O157:H7 was isolated from 0.6% of cattle on 10% of farms, and VTEC was isolated from 46% of the cattle on 100% of farms. Twenty-one people (6.3%) on 16 farms (20.8%) had VTEC in their stools although infection was not associated with diarrheal disease. Of the nine VTEC serotypes found in family members, four were also isolated from cattle on the same farm. The presence of *E. coli* O157:H7 on the farm was associated with antibodies to O157 lipopolysaccharide (LPS) in family members.

A serological study examining VTEC infection in healthy farm family members and urban residents in Ontario showed marked differences between the two groups⁴⁶. Forty-one percent of farm residents had antibodies to Vero toxin 1 and 12.5% had antibodies to O157 LPS, compared to urban residents of Toronto, who had seropositivity rates of 7.7% and 4.7%, respectively. Many farm residents may experience sub-clinical immunising VTEC infections, and exposure to VTEC may have more severe health consequences in visitors to high VTEC areas and in children with declining maternal immunity.

A cluster of infections occurred in a family on an southwestern Ontario dairy farm⁴⁷. The index case was a 13 month old previously healthy boy who was exposed to dairy cattle through direct contact. The child was hospitalized with bloody diarrhea, and *E. coli* O157:H7 (phage type 23, VT 1+, VT 2+) was isolated from his stool. This was the identical strain isolated from calves at this farm and from the boy's older sister, who was asymptomatic. The children had not consumed unpasteurized milk or undercooked beef.

Geographic Study

The geographic distribution of VTEC infections in Ontario has been explored using Geographic Information Systems⁴⁸. In this study, the age-adjusted risk of VTEC infection between 1990 and 1995 was highest in three southern regions of the province, including Bruce and Grey Counties, which corresponded to areas of mixed agriculture and high cattle density. The major urban areas, including Toronto, Ottawa, Hamilton and Niagara, had a

relatively low incidence of infection. This study indicates that the spatial distribution of VTEC incidence is significantly associated with that of cattle density ($R^2=48\%$).

Well Water Study

The relationship between *E. coli* indicator bacterial in well water and gastrointestinal illness in rural families in southern Ontario has been explored⁴⁹. Participating families had been residents of their farm and drinking the well water for at least three years. During the study period, a rate of 0.40 gastrointestinal episodes per person-year was identified. Close proximity of the septic tank to the well was protective against episodes of gastrointestinal illness, and a potential reason may be immunity as a result of a more consistent exposure over time.

SOURCES AND SURVIVAL IN THE ENVIRONMENT

Cattle

In the northern United States, the prevalence of *E. coli* O157:H7 infection in cattle has been shown to range from 1%⁵⁰ to 6.4-9.5%⁵¹. In Ontario, the prevalence of verocytotoxigenic *E. coli* (VTEC) infection is estimated to be 9.5% for cows and 24.7% for calves^{52, 53}, supporting the hypothesis that age affects carriage rate in cattle. The farm-level prevalence of pathogenic *E. coli* ranges from 41% to 50%^{54, 18, 55}, where a prevalence of 41% has been observed in Holsteins in southwestern Ontario⁵⁵. Ontario on-farm infection rates range from 0-60% for cows and 0-100% for calves⁵², and strains of pathogenic *E. coli* have been demonstrated to persist in herds for up to two years⁵¹.

In an Iowa-based study, the observed prevalences of *C. jejuni* and *C. coli* infections in cattle were 38% and 2%, respectively, and the farm-level prevalences of *C. jejuni* and *C. coli* were 81% and 19%, respectively⁵⁶. In southwestern Ontario, 13% of Holstein farms are estimated to be infected with *C. jejuni* or *C. coli*⁵⁵.

Manure

E. coli O157 inoculated into cattle feces remained detectable at high levels for more than 50 days. In contrast, the organism survived much less readily in cattle slurry, where it declined more than 10^6 organisms/gram to reach undetectable levels in 10 days⁵⁷. In a California-based study, the prevalence of *Campylobacter* spp. infection in cattle was found to be 5% for rectal samples and 0.5% for ground fecal samples, suggesting the organism dies off in the feces and that prevalence estimates based on ground fecal samples may provide a more accurate estimate of the burden of the pathogen in the environment⁵⁸.

Soil

Studies indicate that if *E. coli* O157 reaches the soil surface, either through manure application or run-off, it may have the potential to survive, replicate and move vertically over time. Rainfall may result in pathogen spread into the soil, either by runoff from stored or unincorporated manure or by leaching through the soil profile⁵⁹. In one study, transport of *E. coli* and *E. coli* O157 from cattle slurry on the soil surface deeper into soil cores was mainly associated with rainfall events, where transport increased during rainfall events (7% of amount in the slurry was transported) compared to non-rainfall events (1% of amount in slurry was transported)⁵⁷. In another study where growth of the organism in soil was observed, rainfall events were associated with high rates of growth for *E. coli* O157:H7⁵⁹. Saturated soil conditions may enhance growth of facultative anaerobes like *E. coli* O157 within the soil by limiting the amount of oxygen.

Soil characteristics may play a role in the survival of *E. coli*. The presence of manure has been shown to enhance the survival of *E. coli* O157:H7 in no-till soils⁵⁹. As well, survival of *E. coli* O157 in soil cores appears greatest in cores containing rooted grass, where, after 130 days, viable numbers only declined from 10^8 to 10^6 - 10^7 organisms/gram of soil⁵⁷.

Water – Environmental

In surface water aquatic environments, *E. coli* may retain growth potential and metabolic activity⁶⁰, however for what length of time is unknown. In one study, *E. coli* O157 in river water fell more than 10^6 organisms/gram to undetectable levels within 27 days⁵⁷. For *Campylobacter* spp., maximal survival in sterile river water occurred at 5°C (versus 15, 25, and 37°C), and $> 10^4$ cfu / mL was maintained for the 60 day duration of the experiment, regardless of the presence of additional nutrients⁶¹.

General aquifer and groundwater properties that influence microbial transport rates include flow velocity, grain/pore size of the aquifer material, amount of solid organic carbon content, pH, and temperature⁶². *Campylobacter jejuni* has been isolated from groundwater⁶³, and in this study some strains had identical biotypes to those isolated from a dairy farm situated in the hydrologic catchment area of the polluted spring.

Drinking Water and Distribution Systems

E. coli can survive in drinking water for four to twelve weeks depending on environmental conditions⁶⁴. *E. coli* O157:H7 may actually survive better in municipal water versus surface water and may enter a viable but non-culturable state in both municipal and environmental water⁶⁵. Thus, it is suggested that emphasis be put on catchment management to limit contamination of raw water and to ensure that the number of *E. coli* in the source water remain low⁶⁶.

E. coli O157:H7 survives at a rate similar to that of typical *E. coli* strains, suggesting that detection of usual strains could indicate the presence of pathogenic strains⁶⁷. Pathogenic *E. coli* O157 are as easily inactivated by chlorine as non-pathogenic wildtype strains^{68, 69}; however, in one experiment under nutrient-limited conditions, VTEC O157 appeared to develop a chlorine resistant phenotype⁷⁰. Generally, chlorine levels typically maintained in drinking water distribution systems are sufficient to inactivate these organisms⁷¹, although adequate disinfection residuals must be maintained in all areas of the water distribution system¹. Lab strains and animal-based strains of *Campylobacter jejuni* may be more susceptible to disinfection than the control organism *E. coli*⁶⁷, and so disinfection practices commonly used in drinking water treatment should be sufficient to eliminate *C. jejuni*.

E. coli O157:H7 does not survive well in laboratory biofilms and appears to be eliminated from such systems within two weeks⁶⁷. In one study from the United Kingdom, there was no evidence that *E. coli* grew within the water distribution system, including in the biofilm⁶⁶.

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